

Morphologic spectrum of truncal valvar origin relative to the ventricular septum: Correlation with the size of ventricular septal defect

Iki Adachi, MD,^a Anna Seale, MD,^b Hideki Uemura, MD, FRCS,^c Karen P. McCarthy, BS,^a Philip Kimberley,^d and Siew Yen Ho, PhD, FRCPath, FESC^a

Objective: The common arterial trunk usually has a balanced origin from both right and left ventricles overriding a ventricular septal defect. The trunk occasionally originates predominantly, or even exclusively, from either ventricle, making the size of the ventricular septal defect an important factor in surgical repair.

Methods: We examined 56 autopsy specimens and reviewed another series of 12 consecutive patients with the malformation. Truncal origin was categorized as 1 of the following 5 types: exclusive origin from either the right or left ventricle, predominant origin from either ventricle, or balanced origin. We measured the size of ventricular septal defect (“width” and “depth”) in specimens for any correlation with truncal origin.

Results: Balanced origin was seen in approximately one half of cases in both autopsy and clinical series. Predominantly or exclusively right ventricular origin was more prevalent than left ventricular origin in autopsy series (40% vs 9%, respectively), but such predilection was not observed in clinical series (both 25%). The more the truncal valve was committed to the right ventricle, the smaller was the “width” of the ventricular septal defect (predominant and exclusive vs balanced origin; both $P < .0001$), with similar tendency in the “depth.” In 1 heart with extreme right ventricular origin, the defect was slit-like.

Conclusion: Origin of the truncal valve demonstrated a morphologic spectrum and correlated with the size of ventricular septal defect that was the main or even sole exit from the left ventricle in hearts with right ventricular origin. Truncal origin, therefore, requires recognition to optimize surgery. (*J Thorac Cardiovasc Surg* 2009; 138:1283-9)

Common arterial trunk (persistent truncus arteriosus) is characterized by a single arterial vessel arising from the base of the heart, through a common truncal valve, which directly gives rise to the systemic, pulmonary, and coronary arteries.¹ Another key feature of this malformation is the presence of a ventricular septal defect (VSD), which is typically large, located between the limbs of the septo-marginal trabeculation, and situated underneath the truncal valve. The common trunk usually overrides the VSD, with approximately equal commitment to both the right ventricle (RV) and the left ventricle (LV). In some cases,

the common trunk originates predominantly, or even exclusively, from one ventricle or the other.²⁻⁵ Although predominantly LV origin favors anatomic repair for connecting the trunk to the LV, origin of the common trunk primarily from the RV could compromise surgery by making the LV outflow tract more tortuous and elongated. The latter morphology is of further practical importance during surgical repair because such truncal origin occasionally accompanies a restrictive VSD, the main outlet from LV.²⁻⁵ Although some authors concluded this unbalanced truncal origin is a risk factor for early mortality⁶ and late morbidity⁷ after surgical repair, truncal origin has gained little attention in the clinical setting. In fact, truncal origin was not specified and taken into account as a potential risk factor for surgery in any of the major clinical studies conducted on this malformation.⁸⁻¹⁷ The paucity of clinical attention on this issue is in marked contrast with observations made in previous pathologic investigations²⁻⁵ showing that RV origin of the common trunk occurs fairly frequently. This obvious difference might reflect an inherent selection bias in autopsy studies, because such truncal origin could be associated with poorer outcome. Alternatively, it might be an omission in clinical series. With these considerations in mind, we sought to review variations of truncal origin in both autopsy and clinical series, focusing on its possible association with the size of VSD.

From the Cardiac Morphology Unit,^a National Heart and Lung Institute, Imperial College London, London, United Kingdom; Department of Pediatric Cardiology,^b Cardiothoracic Surgery,^c and Clinical Governance,^d Royal Brompton and Harefield NHS Trust, London, United Kingdom.

This study is supported by the Francis Fontan prize of the European Association of Cardiothoracic Surgery awarded to Iki Adachi and a grant from The Uehara Memorial Foundation. The Cardiac Morphology Unit receives funding from the Royal Brompton and Harefield Hospital Charitable Fund.

Disclosures: None.

Received for publication Jan 20, 2009; revisions received March 31, 2009; accepted for publication May 15, 2009.

Address for reprints: Siew Yen Ho, PhD, FRCPath, FESC, Cardiac Morphology Unit, National Heart and Lung Institute, Imperial College London, Guy Scadding Building, Dovehouse Street, London SW3 6LY, United Kingdom (E-mail: yen.ho@imperial.ac.uk).

0022-5223/\$36.00

Copyright © 2009 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2009.05.009

Abbreviations and Acronyms

LV = left ventricle
RV = right ventricle
VSD = ventricular septal defect

MATERIALS AND METHODS

This study was approved by the ethics committee of the Royal Brompton Hospital.

Autopsy Series

From the cardiac specimen archive of the Royal Brompton Hospital, we identified 62 postnatal hearts with common arterial trunk. For the purpose of this study, we excluded 2 hearts in which truncal origin could not be determined because of previous dissection in 1 and previous surgery (truncal valve replacement) in 1. In addition, 4 hearts associated with atrioventricular septal defect were not included in our morphometric analyses because their intracardiac anatomy could not be compared with hearts with a usual atrioventricular junction. Nevertheless, the key feature of these hearts is discussed separately in this article, and more detail will be published elsewhere.¹⁸ The remaining 56 hearts were examined. All the hearts examined had the cardinal feature of a common arterial trunk, namely, an arterial trunk arising from the base of the heart that gave rise to systemic arteries, coronary arteries, and at least 1 pulmonary artery. Accordingly, hearts with the so-called type IV anatomy of the Collet-Edwards classification¹⁹ were not included in this study. All hearts had the usual atrial arrangement (situs solitus) and concordant atrioventricular connections through separate atrioventricular junctions. Of the 56 hearts examined, 17 had evidence of previous surgical intervention; 16 hearts had undergone the Rastelli type repair, and 1 heart had previous banding of the pulmonary trunk. The age at the time of death in these hearts ranged from 3 days to 15 years, with most (88%) of them aged 3 months or less.

Truncal Origin Relative to Ventricular Septum

Truncal origin was categorized into 1 of 5 patterns: exclusively connected to either the RV or LV, predominantly connected to either the RV or LV, or balanced origin (Figure 1). Truncal origin was assessed mainly through viewing the truncal valve from the arterial aspect, supplemented by assessment from right and left ventriculotomies. When the middle of the truncal valve was located just above the crest of VSD, the truncal origin was considered balanced. If the middle of the valve digressed from the crest of VSD rightward or leftward but the valve was still committed to both ventricles, the trunk was regarded as arising “predominantly” from the RV or LV, respectively. When the truncal valve was more displaced so that it was barely or no longer committed to 1 ventricle, it was designated as having “exclusive” origin from the other ventricle. Each heart was examined at

least twice on separate occasions by 2 researchers individually to confirm little intra- and interobserver variability.

Size of Ventricular Septal Defect

The “width” of the VSD was defined as the length of a straight line joining the 2 points where the anterior and posterior limbs of the septomarginal trabeculation (or the ventricular infundibular fold) met the truncal valvar annulus (Figures 1 and 2). The “depth” was defined as the distance of the deepest portion of VSD from the straight line used to measure the “width” (Figure 1). We also measured the truncal valvar diameter along a line parallel to the ventricular septum. The valvar diameter was used to standardize the measurements on VSD. Because the size of the hearts varied within the series, standardization allowed us to make direct comparisons of the VSD size. Each measurement was made twice to minimize potential sources of error from manual maneuvers, and the mean values of the 2 measurements were used as final discrete values.

Clinical Series

We identified all patients with a common arterial trunk whose echocardiographic images were stored in the digital archive system of the Royal Brompton Hospital. There were 12 consecutive patients who had undergone primary 1-stage repair at the Royal Brompton Hospital in the previous 3 years. Age at operation in these patients ranged from 2 days to 6 months with a median of 32 days. Surgical procedures performed involved connection of the LV to the common trunk with a surgical patch through a right ventriculotomy and RV outflow tract reconstruction using either an external conduit (homograft or xenograft) or a direct anastomosis technique, depending on the availability of conduits and surgeon’s preference. No patient had a VSD enlargement or required surgical and catheter-based interventions postoperatively.

We retrospectively reviewed preoperative echocardiographic images of all 12 patients. Echocardiographic studies were performed on an HP 5500 or HP7500 (Hewlett-Packard, Palo Alto, CA) with probes of appropriate frequency (5.0–12MHz). All echocardiographic assessment was carried out using an image viewer (TCS Symphony, ver 2.02. Medcon Inc, Middlesex, UK) by 2 researchers: the primary investigator (IA) and a pediatric cardiologist (AS) who had been blinded to the result of morphometric analyses in the autopsy series. Although all available images were reviewed in every patient, the parasternal long axis cut at end diastole was used for determination of truncal origin. Truncal origin was described using the same 5 categories as in the autopsy series.

Statistical Analysis

Because of the small number of cases with predominantly or exclusively LV origin, intergroup comparisons were performed among the remaining 3 groups, namely, predominantly or exclusively RV origin and balanced origin. The Kruskal–Wallis test with Bonferroni correction was used to determine any difference among the 3 groups. The Mann–Whitney test was used

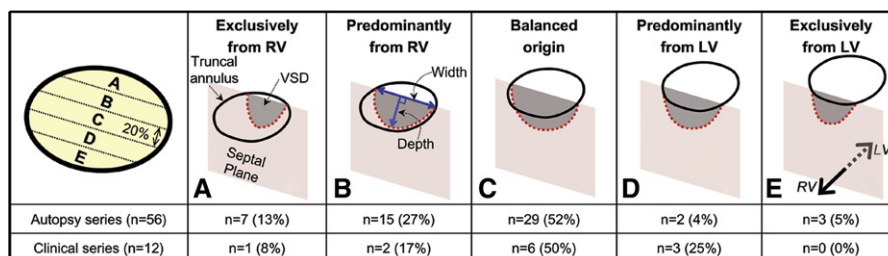


FIGURE 1. Truncal origin was categorized as having 1 of the following 5 types: exclusive origin from either ventricle, predominant origin from either ventricle, or centrally located. The prevalence of each group in both the autopsy and clinical series is shown. B, The measurements made on the VSD to determine the “width” and “depth.” LV, Left ventricle; RV, right ventricle; VSD, ventricular septal defect.

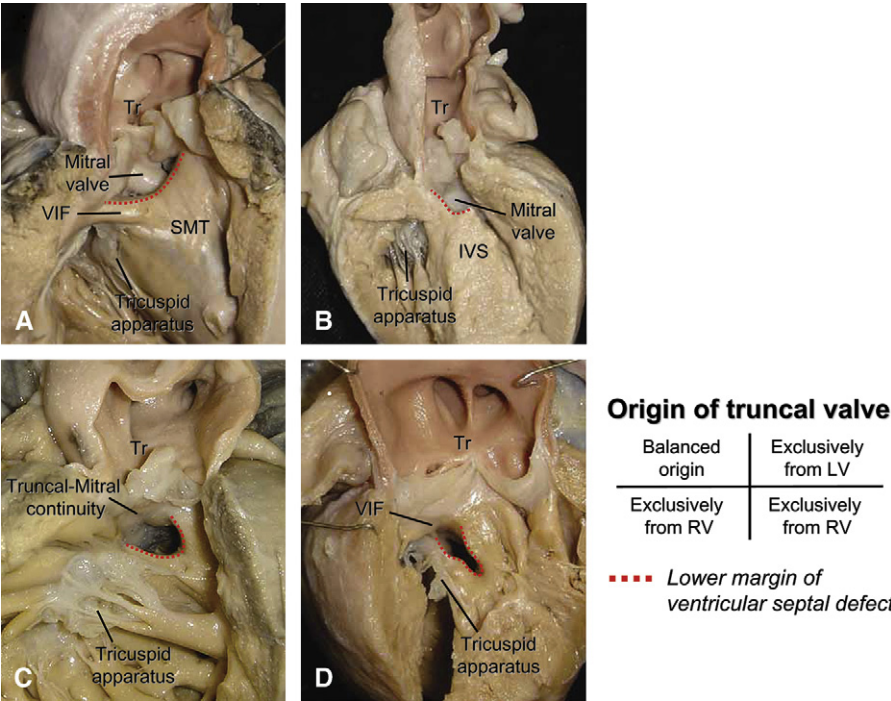


FIGURE 2. Photographs illustrating VSD and its relation to the truncal valve in 4 hearts with the common trunk arising from “center” (A), “exclusively from LV” (B), and “exclusively from RV” (C, D). All photographs except for that in (B) were taken from right ventricular side. The heart with balanced origin (A) has a VSD that is wide in shape compared with others. In the heart (B), the trunk is committed exclusively to the LV. In the heart (C), the truncal–mitral fibrous continuity is still maintained, albeit to a lesser extent than usual. By contrast, the heart (D) has a persistent ventriculo–infundibular fold interposing between the truncal, mitral, and tricuspid valves, creating valvar discontinuity. Consequently, the VSD is no longer “subtruncal.” *IVS*, Interventricular septum; *LV*, left ventricle; *RV*, right ventricle; *VSD*, ventricular septal defect; *Tr*, truncal root; *SMT*, septomarginal trabeculation; *VIF*, ventriculo–infundibular fold.

when comparing 2 groups. The data were analyzed with SPSS 15.0.1 for Windows (SPSS Inc, Chicago, IL).

RESULTS

Autopsy Series

Types of specimens. The origins of the left and right pulmonary arteries were assessed and categorized as from a common orifice (type I according to the Collet-Edwards classification;¹⁹ n = 36), separate and adjacent orifices (type II; n = 13), separate and remote orifices (type III; n = 5), or a single orifice leading to only 1 pulmonary artery (n = 2). The mode of pulmonary origins seemed to have no apparent relationship with the truncal origin or size of the VSD, both of which are described below.

The truncal valve had 2 leaflets in 6 hearts, 3 leaflets in 31 hearts, 4 leaflets in 17 hearts, and 5 leaflets in 1 heart, and could not be assessed in 1 heart. Again, the number of leaflets did not have any obvious association with the truncal origin or VSD size.

Truncal origin. The proportions of each type of truncal origin in the autopsy series are shown in Figure 1. Approximately one half (52%) had a balanced origin (Figure 2). The remaining half had the common trunk committed predominantly or exclusively to 1 ventricle, with RV origin in total

being 4-fold more prevalent than LV origin (40% vs 9%, respectively). Similarly, exclusive RV origin was 2-fold more prevalent than exclusive LV origin (13% vs 5%, respectively) (Figure 2).

Size of ventricular septal defect. Measurements of the VSD size were carried out in 52 of the 56 hearts (93%) examined. In the remaining 4 hearts, it was not possible to assess the VSD size because of either a tightly adherent and endothelialized patch placed at surgical repair or previous dissection and preparation.

Standardized values of the VSD size were plotted according to truncal origin (Figure 3). The width of the VSD showed a clear trend; the more the common trunk deviated from the center either toward right or left, the smaller became the width. The depth showed a similar tendency, but the differences among groups were less obvious than the width, and considerable overlaps were observed among the groups. As a result, the depth/width ratio yielded a reciprocal pattern of those of the width and depth; the more the trunk deviated, the larger became the ratio, suggesting a narrower shape of the defect in hearts with more deviation of the common trunk.

Intergroup comparisons among the 3 groups (predominantly or exclusively RV origin and balanced origin)

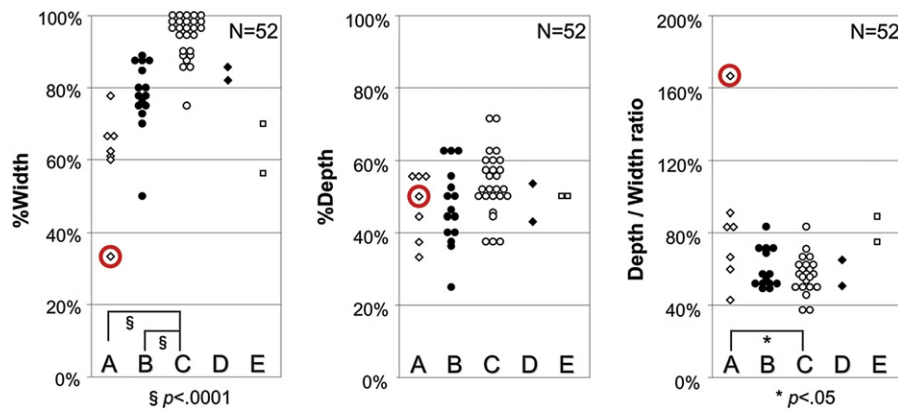


FIGURE 3. Graphs showing the results of measurements on a VSD size according to truncal origin. The “width” and “depth” of the defect are standardized with the truncal valvar diameter of the corresponding heart. The red circle represents a heart with the mitral–truncal fibrous discontinuity. LV, Left ventricle; RV, right ventricle; %width, width/truncal valvar diameter; %depth, depth/truncal valvar diameter.

showed a significant difference in the width ($P < .0001$) and a marginal difference in the depth/width ratio ($P = .043$). The difference in the depth did not reach statistical significance ($P = .113$). When each of the RV origins was compared with balanced origin, the width was significantly smaller in both predominantly ($P < .0001$) and exclusively ($P < .0001$) RV origin than in balanced origin. Similarly, the depth/width ratio was significantly larger ($P = .028$) in exclusively RV origin than in balanced origin. However, this ratio did not differ significantly between predominantly RV origin and balanced origin ($P = .946$).

Fibrous continuity of truncal valve with atrioventricular valves. The morphology of the VSD could be determined in 53 of the 56 hearts (95%) examined. In 43 hearts, fusion between the ventriculo-infundibular fold and the posterior limb of the septomarginal trabeculation produced a muscular postero-inferior margin to the VSD (Figure 2, A). The VSD was perimembranous, and there was a fibrous continuity among the truncal, tricuspid, and mitral valves in all the remaining 10 hearts but 1. Except for this exceptional case, there was always the truncal–mitral fibrous continuity even in hearts with exclusively RV origin, albeit to a lesser extent than usual (Figure 2, C). In this atypical heart, there was a persistent ventriculo-infundibular fold that inserted to the anterior limb of the septomarginal trabeculation, thereby separating the truncal valve from mitral and tricuspid valves, but was attenuated posteriorly, allowing fibrous continuity between the mitral and tricuspid valves (Figure 2, D). Accordingly, its slit-like VSD was considered not only perimembranous but also “noncommitted” to the truncal valve. This peculiar heart appeared to be at the extreme end of the spectrum.

Clinical series. The proportions of each type of truncal origin in the clinical series are shown in Figure 1. The prevalence of balanced origin was almost identical to that in the autopsy series. Unlike the autopsy series, the prevalence of RV origin in total was the same as that of LV origin (both

25%). However, exclusive commitment to 1 ventricle was found only in the RV (1 case). Truncal–mitral fibrous continuity was observed in all 12 patients (Figure 4).

DISCUSSION

In the past, several anatomic features have been considered as risk factors for mortality and morbidity after surgical repair for common arterial trunk. Examples of these conditions include the presence of interrupted aortic arch,^{11,12,17} nature of the truncal valve,^{8,9,11,12,16} and abnormalities of coronary arteries.^{12,16,20} In addition to these anatomic factors, the pattern of pulmonary arterial origin has always gained the greatest attention as considered in the classic Collet and Edwards¹⁹ and Van Praagh²¹ classifications. By contrast, little attention has been directed toward truncal origin relative to the ventricular septum. In fact, truncal origin was not specified or taken into account as a potential risk factor for surgery in any of the large clinical studies conducted on this malformation.⁸⁻¹⁷ If the truncal valve is committed more to LV, such ventriculoarterial connection would work in the surgeon’s favor when aiming to achieve anatomic repair. However, when truncal origin is displaced toward the RV, this particular ventriculoarterial relationship would be potentially problematic because of the elongated and tortuous LV outflow subsequent to surgical repair. This is exacerbated when the VSD is restrictive, because the defect serves as the LV outlet pathway. Although enlargement of the VSD may be an option in this setting, such a maneuver is not risk-free.^{10,22} Trowitzsch and associates⁶ at the Boston Children’s Hospital have concluded that alignment of 60% or more of the truncal root over the RV (predominant or exclusive RV origin in our case) is a strong predictor of postoperative mortality, although their study has been published as an abstract only. To the best of our knowledge, there are no other clinical articles discussing the impact of truncal origin on early outcome after repair. The paucity of articles on this issue has been pointed out in

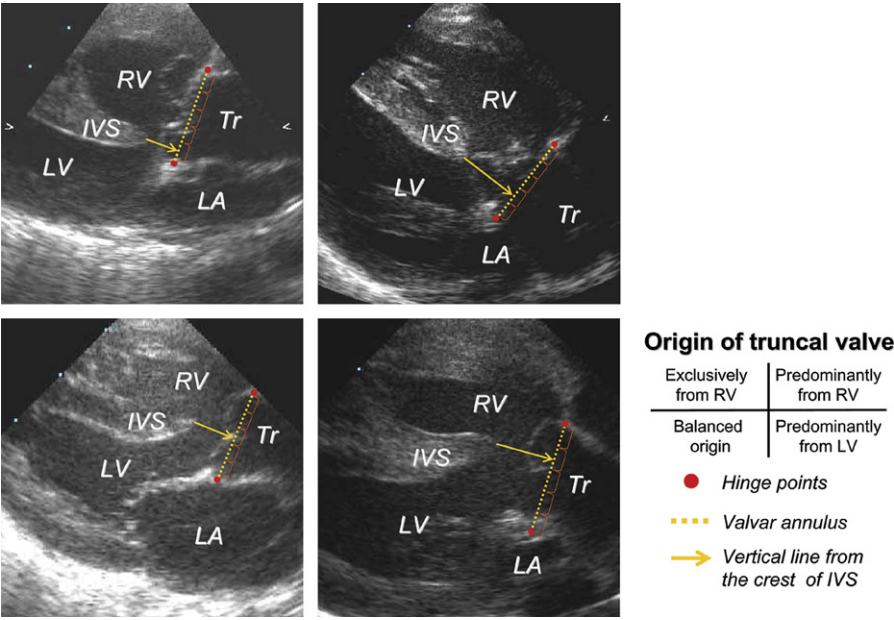


FIGURE 4. Parasternal long-axis echocardiographic images obtained at end diastole from 4 patients in the clinical series with different types of tricuspid origin. The valvar annulus and its hinge points are marked with the yellow dotted line and the red circle, respectively. The valvar diameter is divided into 5 equal portions (notched line), and tricuspid origin is determined by a perpendicular line from the crest of the ventricular septum indicated with the arrow. IVS, Interventricular septum; LA, left atrium; LV, left ventricle; RV, right ventricle; Tr, tricuspid root.

Kirklin/Barratt-Boyes Cardiac Surgery.²³ Furthermore, tricuspid origin could also affect long-term result. Delius and colleagues⁷ described development of functional subtricuspid valvar stenosis 11 years after initial repair in a patient with the trunk mainly originating from RV accompanied with a relatively small VSD. Even if the VSD size is not hemodynamically significant at the time of operation, it could become smaller because of changes in ventricular geometry after complete repair.²⁴

Our morphometric study in the autopsy series has revealed that the common arterial trunk has RV origin more frequently than LV origin (Figure 1). This finding is concordant with the observations in the previous anatomic studies.²⁻⁵ However, there is disagreement among these studies regarding the exact prevalence of each type of origin (Table 1). In particular, the prevalence of balanced origin in the study by Bharati and colleagues² is significantly different from that in other articles. In their study, only a small portion (16.5%) had balanced origin, whereas the majority (66.5%) had the tricuspid origin mostly (predominantly) from the RV.

These figures are in marked contrast with the other articles,³⁻⁵ all of which considered balanced origin as the most common type. A possible explanation would be the difference in the way of assessing the tricuspid origin. In our study, tricuspid origin was assessed mainly through the tricuspid valve rather than through a right ventriculotomy. Another vital issue regarding tricuspid origin is whether the relatively high prevalence of RV origin in the autopsy series can be extrapolated to the whole spectrum of this malformation encountered in the clinical setting. To address this question, we reviewed data on consecutive clinical patients. As a result, although we found a similar proportion of balanced origin, the prevalence of RV origin was not higher than that of LV origin in the clinical series. Furthermore, there seemed to be no extreme case of exclusive RV origin, and fibrous continuity between tricuspid and mitral valves was observed in all of them. Echocardiographic evaluation could be compared with autopsy assessment in only 1 of our patients. In this case, the categorization on autopsy study exactly matched that of echocardiographic assessment

TABLE 1. Prevalence of tricuspid origins in the previous autopsy series

Author's name (total No. of specimens)	Exclusively from RV	Predominantly from RV	Balanced origin	Predominantly from LV	Exclusively from LV
Bharati and colleagues ² (177)	25 (14%)	117 (66%)	29 (16%)	5 (3%)	1 (1%)
Thiene and colleagues ³ (12)	3 (25%)		9 (75%)*		0
Crupi and colleagues ⁴ (66)	2 (3%)	7 (11%)	53 (80%)	4 (6%)	0
Butto and colleagues ⁵ (54)		23 (42%)†	23 (42%)	8 (15%)	

LV, Left ventricle; RV, right ventricle. *Described as “biventricular origin.” †Described as “60%–100% origin from RV.”

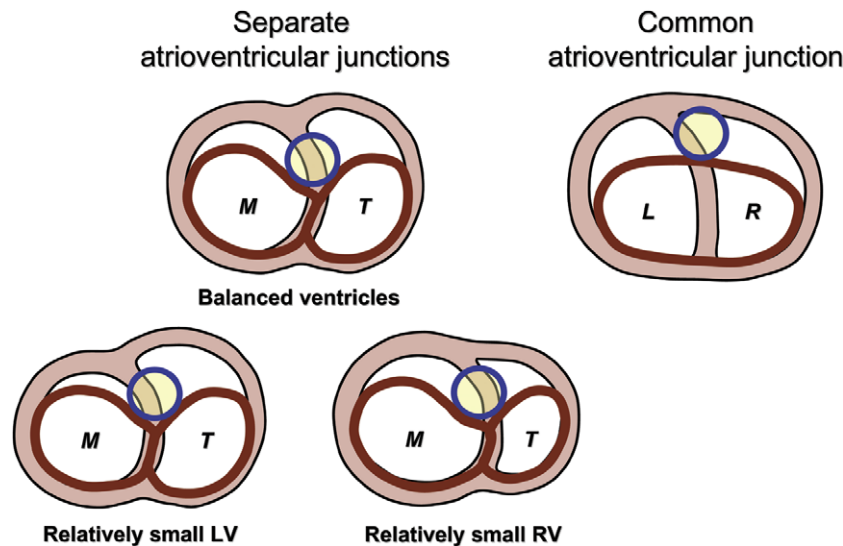


FIGURE 5. Schematic illustrations of the ventricles and atrioventricular junctions in cross-section show the relationship of the truncal valve to the ventricular septum. Any imbalance of the ventricular volume might cause change in the ventriculoarterial relationship owing to the digression of the ventricular septum. Similarly, location of the truncal valve itself could alter this relationship. In hearts with common atrioventricular junction, the anterior location of the truncal valve results in RV origin of the truncal valve. *M*, Mitral valve; *T*, tricuspid valve; *L*, left atrioventricular valve; *R*, right atrioventricular valve; *LV*, left ventricle; *RV*, right ventricle.

(predominantly from the LV). By accepting the small number of our clinical cases, it would be reasonable to say that a common trunk arising from the RV, albeit to a greater or lesser extent, is not an exceedingly rare condition even in the clinical setting.

We measured the VSD size in the autopsy series and looked for a possible relation to truncal origin. Consequently, we found an interesting tendency. The more the truncal valve deviated from its central position of override, the smaller became the width of the VSD. This trend, in accordance with geometry, can be explained by illustrations in Figure 1. This tendency would be of paramount importance for clinical decision making, because it should obviously indicate that hearts with exclusively RV origin are more prone to LV outflow obstruction and thus more likely to require extensive surgery, including enlargement of the VSD. VSD enlargement risks injury to the septal coronary perforators, which are known as additional blood sources to the atrioventricular junctional area²⁵ and are the dominant or even absolute blood supplier for the penetrating conduction bundle or bundle branches.²² Barbero-Marcial and associates¹⁰ reported 1 case of sudden irreversible cardiac arrest and death after common arterial truncal repair in which the restrictive VSD was enlarged. Autopsy revealed necrosis of the His bundle and its 2 branches, whereas the atrioventricular node and its artery were intact. Even if the VSD is adequately enlarged without any complications, we anticipate that hearts with exclusively RV origin would still not be free from solicitude for biventricular function. Owing to a large, bulging patch tunneling within the RV cavity, the RV volume would be inevitably reduced. The LV func-

tion could also be affected by an elongated and tortuous LV outflow tract that would pose a potential nidus for obstruction as seen after the Rastelli-type procedure for other conotruncal anomalies.²⁶

A question that came to our mind is what makes the variations in truncal origin. In other words, what kind of anatomic features should alert clinicians regarding the possibility of such unfavorable truncal origin? Because the ventriculoarterial connection is a mutual relationship between the 2 neighboring structures, it could be affected not only by the truncal valve itself but also by the arrangement of the ventricular mass. Some unusual cases gave us clues for a better understanding on this matter. If the ventricular septum is displaced toward either side for any reason, the common trunk would become committed more to the contralateral ventricle unless there is a concomitant shift of the common trunk toward the same side as the septum. Any imbalance between the 2 ventricles could potentially cause this type of septal displacement (Figure 5). Our hypothesis might be supported by the reported cases of common arterial trunk arising exclusively from the RV in the setting of a hypoplastic LV^{27,28} and their opposite pattern with the trunk exclusively from the LV in the setting of a hypoplastic RV.^{29,30} Second, the location of the truncal valve could have more direct influence on the relationship. As described above, there were 4 specimens with a common atrioventricular junction in the Brompton archive. In all 4 hearts the trunk was exclusively from the RV and the truncal valve was located more anteriorly than in hearts with normal atrioventricular junctions because of the lack of recess that can accommodate the truncal valve (Figure 5). This anterior

location of the truncal valve must make it committed more to the RV, especially because the RV is a more anterior structure than its counterpart.

A limitation of the current study is the small number of cases in our clinical series. By combining our study with a larger autopsy series, we believe our findings are reasonably representative of the whole spectrum of this malformation. Nevertheless, we hope our study will stimulate further clinical investigations on this issue.

CONCLUSIONS

The current study demonstrated that origin of the common trunk represents a morphologic spectrum and correlates with the size of the VSD. Because the VSD is the only exit from the LV, it is important to take note of its size especially when the truncal origin is predominantly or exclusively from the RV. Assessment of truncal origin preoperatively should be routine practice. Moreover, when the common trunk is found to have RV origin, the absence of truncal–mitral fibrous continuity would warrant further clinical attention, because it might represent an extreme end of the spectrum.

The authors express appreciation for statistical advice from Joseph Eliahoo at the Statistical Advisory Service, Imperial College London. We also appreciate Manveer Sroya and Carina Lim for technical and secretarial assistance.

References

1. Ho SY. Common arterial trunk. In: Ho SY, Rigby ML, Anderson RH, eds. *Echocardiography in Congenital Heart Disease Made Simple*. London: Imperial College Press; 2005:164-74.
2. Bharati S, McAllister HA Jr, Rosenquist GC, Miller RA, Tautoles CJ, Lev M. The surgical anatomy of truncus arteriosus communis. *J Thorac Cardiovasc Surg*. 1974;67:501-10.
3. Thiene G, Bortolotti U, Gallucci V, Terribile V, Pellegrino PA. Anatomical study of truncus arteriosus communis with embryological and surgical considerations. *Br Heart J*. 1976;38:1109-23.
4. Crupi G, Macartney FJ, Anderson RH. Persistent truncus arteriosus. A study of 66 autopsy cases with special reference to definition and morphogenesis. *Am J Cardiol*. 1977;40:569-78.
5. Butto F, Lucas RV Jr, Edwards JE. Persistent truncus arteriosus: pathologic anatomy in 54 cases. *Pediatr Cardiol*. 1986;7:95-101.
6. Trowitzsch E, Sluymans T, Parness IA, et al. Anatomy and surgical outcome in infants with truncus arteriosus. *J Am Coll Cardiol*. 1991;17:110A [abstract].
7. Delius RE, Embrey RP, Behrendt DM. Late development of functional subvalvar stenosis after repair of truncus arteriosus. *Pediatr Cardiol*. 1996;17:393-5.
8. Ebert PA, Turley K, Stanger P, Hoffman JI, Heymann MA, Rudolph AM. Surgical treatment of truncus arteriosus in the first 6 months of life. *Ann Surg*. 1984;200:451-6.
9. Di Donato RM, Fyfe DA, Puga FJ, et al. Fifteen-year experience with surgical repair of truncus arteriosus. *J Thorac Cardiovasc Surg*. 1985;89:414-22.
10. Barbero-Marcial M, Riso A, Atik E, Jatene A. A technique for correction of truncus arteriosus types I and II without extracardiac conduits. *J Thorac Cardiovasc Surg*. 1990;99:364-9.
11. Pearl JM, Laks H, Drinkwater DC Jr, et al. Repair of truncus arteriosus in infancy. *Ann Thorac Surg*. 1991;52:780-6.
12. Hanley FL, Heinemann MK, Jonas RA, et al. Repair of truncus arteriosus in the neonate. *J Thorac Cardiovasc Surg*. 1993;105:1047-56.
13. Bove EL, Lupinetti FM, Pridjian AK, et al. Results of a policy of primary repair of truncus arteriosus in the neonate. *J Thorac Cardiovasc Surg*. 1993;105:1057-65.
14. Lacour-Gayet F, Serraf A, Komiya T, et al. Truncus arteriosus repair: influence of techniques of right ventricular outflow tract reconstruction. *J Thorac Cardiovasc Surg*. 1996;111:849-56.
15. Williams JM, de Leeuw M, Black MD, Freedom RM, Williams WG, McCrindle BW. Factors associated with outcomes of persistent truncus arteriosus. *J Am Coll Cardiol*. 1999;34:545-53.
16. Danton MH, Barron DJ, Stumper O, et al. Repair of truncus arteriosus: a considered approach to right ventricular outflow tract reconstruction. *Eur J Cardiothorac Surg*. 2001;20:95-103.
17. Konstantinov IE, Karamlou T, Blackstone EH, et al. Truncus arteriosus associated with interrupted aortic arch in 50 neonates: a Congenital Heart Surgeons Society study. *Ann Thorac Surg*. 2006;81:214-22.
18. Adachi I, Ho SY, Bartelings MM, McCarthy KP, Seale A, Uemura H. Common arterial trunk with atrioventricular septal defect: new observations pertinent to repair. *Ann Thorac Surg*. 2009;87:1495-9.
19. Collett RW, Edwards JE. Persistent truncus arteriosus; a classification according to anatomic types. *Surg Clin North Am*. 1949;29:1245-70.
20. Lenox CC, Debich DE, Zuberhuhler JR. The role of coronary artery abnormalities in the prognosis of truncus arteriosus. *J Thorac Cardiovasc Surg*. 1992;104:1728-42.
21. Van Praagh R, Van Praagh S. The anatomy of common aorticopulmonary trunk (truncus arteriosus communis) and its embryologic implications. A study of 57 necropsy cases. *Am J Cardiol*. 1965;16:406-25.
22. Hosseinpour AR, Anderson RH, Ho SY. The anatomy of the septal perforating arteries in normal and congenitally malformed hearts. *J Thorac Cardiovasc Surg*. 2001;121:1046-52.
23. Kouchochos N, Blackstone EH, Doty DB, Hanley FL, Karp RB. Truncus arteriosus. In: Kirklin JW, Barratt-Boyes BG, eds. *Cardiac surgery*. 3rd ed, vol 2. Philadelphia: Elsevier; 2003:1220-1.
24. Rychik J, Jacobs ML, Norwood WI. Early changes in ventricular geometry and ventricular septal defect size following Rastelli operation or intraventricular baffle repair for conotruncal anomaly. A cause for development of subaortic stenosis. *Circulation*. 1994;90(5 Pt 2):II13-9.
25. Frink RJ, James TN. Normal blood supply to the human His bundle and proximal bundle branches. *Circulation*. 1973;47:8-18.
26. Rocchini AP, Rosenthal A, Castaneda AR, Keane JF, Jersaty R. Subaortic obstruction after the use of an intracardiac baffle to tunnel the left ventricle to the aorta. *Circulation*. 1976;54:957-60.
27. Alves PM, Ferrari AH. Common arterial trunk arising exclusively from the right ventricle with hypoplastic left ventricle and intact ventricular septum. *Int J Cardiol*. 1987;16:99-102.
28. Murdison KA, McLean DA, Carpenter B, Duncan WJ. Truncus arteriosus communis associated with mitral valve and left ventricular hypoplasia without ventricular septal defect: unique combination. *Pediatr Cardiol*. 1996;17:322-6.
29. Sharma D, Mehta AB, Bharati S, Lev M. Tricuspid atresia with persistent truncus arteriosus. *Chest*. 1981;79:363-5.
30. Malec E, Mroczek T, Pajak J, Kordon Z. Operative treatment of truncus arteriosus communis coexisting with tricuspid atresia. *Ann Thorac Surg*. 2000;69:278-80.